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EXAMINER	
VIVLEMORE, TRACY ANN	
ART UNIT	**PAPER NUMBER**
1635	

DATE MAILED: 04/18/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/028,415	LASHAM ET AL.	
	Examiner	Art Unit	
	Tracy Vivlemore	1635	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on *02 February 2005*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-6,9-14 and 17-27* is/are pending in the application.

4a) Of the above claim(s) *1-5,12,13 and 17-23* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *6,9-11,14 and 24-27* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Priority

Receipt is acknowledged of papers submitted under 35 U.S.C. 119(a)-(d), which papers have been placed of record in the file.

Claim Rejections - 35 USC § 112

Claims 6, 9, 14 and 25 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a written description rejection.

1. Claim 6 is directed to a method of increasing apoptotic cell death in a population of cells by reducing the amount of a transcriptional regulator of apoptosis (hereafter referred to as TRA) available to bind to a target polynucleotide. The TRA is defined to be the human Y-box 1 protein (YB-1) or the cold shock domain of this protein as defined by sequences 40 and 39, respectively. Claim 9 limits claim 6 to use of the claimed method in tumor cells. Claim 14 is directed to a method of increasing p53-mediated

apoptosis by reducing the amounts of a TRA available to bind to a target polynucleotide. Claim 25 limits claim 14 to use of the claimed method in tumor cells.

2. The specification teaches in example 2 (pages 26-29) use of ten antisense and two decoy oligonucleotide sequences to modulate the binding of YB-1 and Pur-α to the CD95 promoter and thus modulate apoptosis in HepG2 (liver carcinoma) cells. In example 4 (pages 30-33) the disclosed antisense and decoy oligonucleotides of YB-1 were also shown to modulate apoptosis by activating p53 in several human cancer cell lines.

3. The instant claims encompass the use of any compound that is capable of reducing the amount of a TRA. Such compounds include antisense or decoy oligonucleotides as well as other types of nucleic acids such as ribozymes or short interfering RNAs as well as non-nucleic acid inhibitors such as antibodies, other protein inhibitors and small organic molecules. The specification discloses antisense and decoy oligonucleotide sequences directed to SEQ ID NO: 40, but there is no description found in the specification or known in the prior art of the structure of compounds such as antibodies or small organic molecules that would serve to inhibit SEQ ID NOS: 39 and 40. The specification lacks adequate description of the structure of inhibitors other than antisense or decoy oligonucleotides to YB-1 or its cold shock domain that would be effective in increasing apoptosis.

4. Vas-Cath Inc. v. Mahurkar, 19 USPQ2d 1111, makes clear that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession *of the invention.* The invention is, for purposes of the 'written description' inquiry, *whatever is now claimed.*" (See page 1117.) The

specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed." (See Vas-Cath at page 1116.)

5. MPEP 2163 states in part, "An adequate written description of a chemical invention also requires a precise definition, such as by structure, formula, chemical name, or physical properties, and not merely a wish or plan for obtaining the chemical invention claimed. See, e.g., *Univ. of Rochester v. G.D. Searle & Co.*, 358 F.3d 916, 927, 69 USPQ2d 1886, 1894-95 (Fed. Cir. 2004) (The patent at issue claimed a method of selectively inhibiting PGHS-2 activity by administering a non-steroidal compound that selectively inhibits activity of the PGHS-2 gene product, however the patent did not disclose any compounds that can be used in the claimed methods. While there was a description of assays for screening compounds to identify those that inhibit the expression or activity of the PGHS-2 gene product, there was no disclosure of which peptides, polynucleotides, and small organic molecules selectively inhibit PGHS-2. The court held that "[w]ithout such disclosure, the claimed methods cannot be said to have been described.")."

6. The skilled artisan cannot envision the detailed structure of the full scope of the encompassed compounds that would reduce the amount of a TRA, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it. See Fiers v. Revel, 25 USPQ2d 1601, 1606 (CAFC 1993) and Amgen Inc. V. Chugai Pharmaceutical Co. Ltd., 18 USPQ2d 1016. In Fiddes v. Baird, 30 USPQ2d 1481, 1483, claims directed to mammalian FGF's were found

unpatentable due to lack of written description for the broad class. The specification provided only the bovine sequence.

7. Therefore, the full breadth of the claimed genus of compounds that can reduce the amount of a TRA and function to increase apoptosis meets the written description provision of 35 USC 112, first paragraph. The species specifically disclosed are not fully representative of the genus because the genus is highly variant. Applicant is reminded that Vas-Cath makes clear that the written description provision of 35 USC 112 is severable from its enablement provision. (See page 1115.)

Response to Arguments

8. Applicant's amendment of February 2, 2005 limiting the transcriptional regulator of apoptosis to SEQ ID NOS: 39 and 40 has removed the grounds for a written description rejection against claims 10 and 11. With the narrower scope removing embodiments directed to antisense inhibition of sequences not fully defined by the specification, Applicant's arguments that the disclosure in the specification is sufficient to describe inhibitors of SEQ ID NOS: 39 and 40 that are antisense and decoy oligonucleotides is persuasive. However, claims 6 and 14 encompass the use of inhibitors of the claimed sequences that are not antisense or decoy oligonucleotides, as stated in the original rejection at paragraph 11 and described in more detail in this Action.

9. Applicant states they are unaware of a requirement in patent law that inventors provide a description of every possible way to carry out a claimed method and states that the specification provides a description of the claimed methods and exemplary

antisense and decoy oligonucleotides. The examiner is also unaware of a requirement that all possible ways of carrying out a claimed method be described but there is a well-established requirement that the specification of a patent provide a description of a sufficient number of species within a claimed genus that would allow one of skill in the art to recognize the inventor had possession of the claimed invention at the time of filing. The exemplary antisense and decoy oligonucleotides are sufficient to establish the inventor had possession of the claimed invention to the extent that the method uses such oligonucleotides as inhibitors of YB-1. However, the claims encompass non-nucleic acid inhibitors such as antibodies or small molecules and the specification provides no description of such inhibitors.

10. Applicant responds to the Examiner's citation of Univ. of Rochester v. G.D. Searle & Co. and Fiddes v. Baird by stating that in contrast to these cases that do not describe any of the claimed compounds the instant specification provides several examples of oligonucleotides that can be employed in the claimed methods. The Examiner concurs that the specification contains a disclosure of several exemplary oligonucleotides, citation of these cases was not meant to imply otherwise. However, these arguments are not persuasive because as stated in the instant rejection, the claimed methods encompass use of large genus of compounds including non-nucleic acid inhibitors of YB-1. The disclosure of several nucleic acid inhibitors that can be employed in the claimed method and the existence of YB-1 antisense plasmids are not sufficient to describe the full genus of compounds encompassed by the claims.

Claim Rejections - 35 USC § 112

Claims 6, 9-11, 14 and 24 are maintained as rejected and new claims 25-27 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for increasing apoptotic cell death *in vitro* and *ex vivo* in cell culture and *in vivo* in the mouse, does not reasonably provide enablement for increasing apoptotic cell death by reducing the amount of a transcriptional regulator of apoptosis available to bind to a target polynucleotide *in vivo* in any other organism. Moreover, the specification does not reasonably provide enablement for a method for treating a disease or infection in an organism. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

The following factors as enumerated *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988), are considered when making a determination that a disclosure is not enabling: the breadth of the claims, the nature of the invention, the state of the prior art, the level of ordinary skill in the art, the level of predictability in the art, the amount of direction provided by the inventor, the existence of working examples and the quantity of experimentation needed to make the invention based on the content of the disclosure.

11. Claims 6, 9-11, 14 and 24-27 are drawn to methods of increasing apoptotic cell death in a population of cells by reducing the amount of a transcriptional regulator of apoptosis (hereafter referred to as TRA) available to bind to a target polynucleotide. The method as claimed encompasses use of inhibitors of a TRA *in vivo* in all organisms. The methods as claimed also encompass use of the method to treat disease, as evidenced by claim 10, which is drawn to the use of the claimed method in tumor cells.

12. The specification teaches delivery methods for therapeutic nucleic acids on pages 14 and 15. The specification teaches in example 2 (pages 26-29) use of antisense and decoy oligonucleotides to modulate the binding of YB-1 and Pur-α to the CD95 promoter and thus modulate apoptosis in HepG2 (liver carcinoma) cells. In example 4 (pages 30-33) antisense and decoy oligonucleotides of YB-1 were also shown to modulate apoptosis by activating p53 in several human cancer cell lines. Example 3 (page 30) describes one *in vivo* use of antisense and decoy oligonucleotides targeted against YB-1 to modulate apoptosis in mice.

13. The state of the prior art is such that use of nucleic acids to modulate cellular processes *in vitro* is well-known, but use of nucleic acids *in vivo* as therapeutic agents at the time of filing and even to the present time is not routine for several reasons, including the problems of delivery, specificity and duration.

14. The problems of nucleic acid based therapies and antisense technology are well known in the art, particularly with regard to the inability to specifically deliver an effective concentration of a nucleic acid to a target cell, such that a target gene is inhibited to a degree necessary to result in a therapeutic effect. For example, at the time the instant invention was made, the therapeutic use of nucleic acids was a highly unpredictable art due to obstacles that continue to hinder the therapeutic application of nucleic acids *in vivo* (whole organism) (see for example Agrawal et al. (Molecular Medicine Today, 2000, vol 6, p 72-81), Branch (TIBS 1998, vol. 23, p. 45-50) and Jen et al. (Stem Cells 2000, Vol. 18, p 307-319)). Such obstacles include, for example, problems with delivery, target accessibility and the potential for unpredictable nonspecific effects.

15. Jen et al. state (see page 313, second column, second paragraph) "One of the major limitations for the therapeutic use of AS-ODNS and ribozymes is the problem of delivery....presently, some success has been achieved in tissue culture, but efficient delivery for *in vivo* animal studies remains questionable". Jen et al. outlines many of the factors limiting the application of antisense for therapeutic purposes and concludes (see p 315, second column), "Given the state of the art, it is perhaps not surprising that effective and efficient clinical translation of the antisense strategy has proven elusive."

16. Opalinska et al. (Nature Review, 2002, vol 1, p. 503-514) state "[I]t is widely appreciated that the ability of nucleic-acid molecules to modify gene expression *in vivo* is quite variable, and therefore wanting in terms of reliability. Several issues have been implicated as a root cause of this problem, including molecule delivery to targeted cells and specific compartments within cells and identification of sequence that is accessible to hybridization in the genomic DNA or RNA" and in column 2 of the same page, "Another problem in this field is the limited ability to deliver nucleic acids into cells and have them reach their target. Without this ability, it is clear that even an appropriately targeted sequence is not likely to be efficient. As a general rule, oligonucleotides are taken up primarily through a combination of adsorptive and fluid-phase endocytosis. After internalization, confocal and electron microscopy studies have indicated that the bulk of the oligonucleotides enter the endosome-lysosome compartment, in which most of the material becomes either trapped or degraded."

17. Given this unpredictability, the skilled artisan would require specific guidance to practice the claimed methods *in vivo* in all organisms, with a resultant modulation of apoptosis as claimed. The specification provides examples of modulation of apoptosis

by targeting of YB-1 in several cell lines, including human cell lines, however, cell culture examples are generally not predictive of *in vivo* inhibition and the methods of delivery of the exemplified cell line would not be applicable to delivery of oligonucleotides to any organism. Often formulations and techniques for delivery *in vitro* (cell culture) are not applicable *in vivo* (whole organism) (see for example Jen et al., page 313, second column, second paragraph). For example, Agrawal et al. (see p 79-80, section entitled "Cellular uptake facilitators for *in vitro* studies") states "The cellular uptake of negatively charged oligonucleotides is one of the important factors in determining the efficacy of antisense oligonucleotides.....*In vitro*, cellular uptake of antisense oligonucleotides depends on many factors, including cell type, kinetics of uptake, tissue culture conditions, and chemical nature, length and sequence of the oligonucleotide. Any one of these factors can influence the biological activity of an antisense oligonucleotide." Due to differences in the physiological conditions of a cell *in vitro* versus *in vivo*, the uptake and biological activity observed *in vitro* would not predictably translate to *in vivo* results. The specification provides one example of antisense and decoy oligonucleotides to YB-1 being delivered to mice via injection, but this example is not predictive of efficacy in any other organism, including humans.

18. Given these teachings, the skilled artisan would not know *a priori* whether introduction of antisense or decoy oligonucleotides *in vivo* by the broadly disclosed methodologies of the instant invention, would result in successful modulation of apoptosis by reducing the amount of a TRA available to bind a target polynucleotide. One of skill in the art would not know how to deliver oligonucleotides to an organism in

such a way that would ensure an amount sufficient to modulate apoptosis is delivered to the proper cell.

19. In fact, the state of the art is such that successful delivery of oligonucleotide sequences *in vivo* or *in vitro*, such that the polynucleotide or oligonucleotide provides the requisite biological effect to the target cells/tissues/organs, must be determined empirically. Methods of inhibiting gene expression using nucleic acids *in vivo* are unpredictable with respect to delivery of the nucleic acid molecule such that the nucleic acid molecule is targeted to the appropriate cell/organ, at a bioeffective concentration and for a period of time such that the nucleic acid molecule is effective in, as in the instant application, attenuating or inhibiting expression of a target gene such that the organism exhibits a loss of function phenotype.

20. The specification does not provide the guidance required to overcome the art-recognized unpredictability of using antisense or decoy oligonucleotides in therapeutic applications in any organism. The field of nucleic acid therapeutics does not provide that guidance, such that the skilled artisan would be able to practice the claimed therapeutic methods.

21. Thus, while the specification is enabling for the examples set forth in the specification, the specification is not enabling for the broad claims of modulating apoptosis in all organisms as the art of introducing nucleic acids into an organism for therapeutic applications is neither routine nor predictable. In order to practice the claimed invention *in vivo* in all organisms a number of variables would have to be optimized, including 1). the form of the antisense or decoy oligonucleotide, whether to use a modified oligonucleotide with one or more backbone, sugar or base modifications,

2). the mode of delivery of the antisense or decoy oligonucleotide to an organism that would allow it to reach the targeted cell, 3). the amount of antisense or decoy oligonucleotide that would need to be delivered in order to bind a sufficient amount of YB1 to modulate apoptosis once it reached the proper cell and 4). ensuring the antisense or decoy oligonucleotide remains viable in a cell for a period of time that allows modulation of apoptosis to an extent that there is a measurable and significant therapeutic effect. Each one of these variables would have to be empirically determined for each antisense or decoy oligonucleotide. While optimization of any single one of these steps may be routine, when taken together the amount of experimentation required becomes such that one of skill in the art could not practice the invention commensurate in scope with the claims without undue, trial and error experimentation and therefore, claims 6, 9-11, 14 and 24-27 are not enabled.

Response to Arguments

22. Applicant has argued that the example of use of an antisense oligonucleotide described in the specification is enabling for the claimed methods which encompass *in vivo* treatment of all animals with inhibitors of a TRA. In support of this argument Applicant discusses two examples where mouse models have been used as model organisms for testing of drugs. Hudziak et al. and Baselja et al. discuss the testing in both cells and mouse models of the drug Herceptin, which is an antibody that inhibits the HER2 protein. However, these references do not relate to the YB-1 gene and provide no evidence that inhibition of HER2 would translate to inhibition of the YB-1 gene, not HER2. Webb et al. discuss an antisense drug to BCL-2 but do not discuss

inhibition of YB-1, providing no evidence that similar results can be expected. The arguments citing these references are not persuasive because the use of mice as models for testing of drugs directed to genes different from that targeted by the claimed method does not demonstrate that the field of nucleic acid therapeutics is predictable.

23. These arguments are also not persuasive because the rejection of record is not concerned with the question of whether mice are a suitable model organism for preclinical testing, but with whether a level of skill exists in the art wherein it is recognized that delivery of a nucleic acid can be done in a predictable fashion. Delivery is more than simple administration: injection of an antisense into the bloodstream of an animal does not guarantee that the drug will reach and enter the cell. Cellular uptake is an important question, as stated by Agrawal. Without a predictable delivery method it is impossible to know how much to administer. If injection will result in the drug dispersal throughout the circulatory system, how much will reach the actual diseased cell? In order to have a therapeutic effect, a drug has to get into a diseased cell in a sufficient concentration and remain viable for a sufficient length of time to affect the expression of the target gene.

24. The references that have been cited provide evidence that methods of delivering nucleic acids to an organism such that the nucleic acid is targeted to and enters the proper cell in a sufficient concentration and for a sufficient period of time to have a therapeutic effect were not predictable at the time of filing of the instant invention and remain unpredictable to the present time. Applicant's arguments regarding the use of mice as model organisms do not persuasively address these issues.

Claim Rejections - 35 USC § 102

25. The rejection of claims 6, 9, 11 and 24 as being anticipated by or obvious over Wada et al. is withdrawn. Applicant's arguments that the claimed method requires functional p53 combined with references demonstrating this protein is non-functional in the cell line used by Wada et al. is persuasive.

Claims 6, 9 and 10 are maintained as rejected under 35 U.S.C. 102(b) as being anticipated by Ohga et al. (Cancer Research, 1996, vol 56, pages 4224-4228).

26. Claim 6 is drawn to a method of increasing apoptotic cell death in a population of cells by reducing the amount of a transcriptional regulator of apoptosis (TRA) available to bind to a target polynucleotide. The TRA is defined to be the human Y-box 1 protein or the cold shock domain of this protein as defined by sequences 40 and 39, respectively. Claim 9 limits claim 6 by stating the cells are tumor cells. Claim 10 limits claim 6 by stating the method is performed by contacting the cells with an antisense oligonucleotide targeted to the TRA.

27. Ohga et al. disclose that KB cells (a tumor cell line) that are transfected with an expression vector containing a sequence that is antisense to YB-1 have a reduced level of the YB-1 protein. (see abstract and p. 4226, second column, under heading "Expression of YB-1 Antisense RNA and Drug Sensitivity") Thus, they have reduced amount of the TRA YB-1 in a tumor cell with an antisense oligonucleotide. Ohga et al. do not disclose that apoptosis has been increased in these cells, but as they disclose all the steps of the method of claim 6, it would be inherent in the cells of Ohga et al. that apoptosis is increased.

28. Thus, Ohga et al. disclose the method of claim 6 and all limitations of claims 9 and 10.

Response to Arguments

29. Applicant's arguments filed February 2, 2005 with regard to the prior art rejections over Ohga et al. have been fully considered but they are not persuasive. Applicant argues that Ohga et al. does not anticipate or render obvious the instant claims because the KB cell line used is not well-characterized and probably has a compromised p53 pathway. These arguments are not persuasive because no factual evidence supporting the assertion that the p53 pathway is compromised in this cell line has been provided.

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Tracy Vivlemore whose telephone number is 571-272-2914. The examiner can normally be reached on Mon-Fri 8:45-5:15.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's acting supervisor, Andrew Wang can be reached on 571-272-0811. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance.

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April 6, 2005

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